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FOR THE AWARD OF DEGREE OF

MASTER OF SURGERY

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Clinical study and management of secondary glaucoma

by

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List of Abbreviations

AC	Anterior chamber
HPMC	Hydroxypropyl methylcellulose
ICE	Iridocorneal Endothelial Syndrome
i.e.	That is
IOP	Intra ocular presssure
LIG	lens induced glaucoma
No.	Number
NVG	Neovascular glaucoma
OD	Once daily
OPD	Out patient department
Pigment Disp	Pigment dispersion syndrome
PXG	Pseudo exfoliation glaucoma
PXS	Pseudo exfoliation syndrome
Tab	Tablet
WHO	World health organization
yrs	Years
%	Percentage
MSICS	Manual small incision cataract surgery
UGH	Uveitis-Glaucoma-Hyphema

Introduction

Glaucoma is a chronic, progressive optic neuropathy caused by a group of eye diseases that lead to damage to the optic nerve head and a change in visual field. The most common risk factor generally acknowledged is a rise in intraocular pressure.[1]
Primary or secondary glaucoma are the two categories. Primary glaucoma is impacted by genetics. Secondary glaucoma occur when an underlying main ocular or systemic disease causes the IOP to increase.

Quite a few times secondary glaucoma goes undiagnosed or even if it is diagnosed it's treatment varies with each type of glaucoma. Consequently, early detection and treatment are crucial to preventing vision deterioration.
The main cause of permanent blindness in the globe is glaucoma. According to WHO over 7.7 million persons with glaucomatous optic neuropathy are thought to exist in the globe.[2] By 2040, it is estimated that there will be 111.8 million cases of glaucoma worldwide.[3]

In a recent study done in Aligarh of 680 total patients overall prevalence of glaucoma was found to be 5%, of which secondary glaucoma was 1% .[4] Secondary glaucoma is divided into secondary open angle and secondary angle closure and these are further divided by basis of different mechanisms of causing secondary glaucoma

According to a study of 7079 patients conducted in Nepal on Glaucoma patients, 528 had secondary glaucoma i.e. 7% of the total cases. Out of these the most common was Lens-induced 33% then came Neo-vascular 20% followed by steroid-induced 16%, Trauma-induced 14%, Post-Vitrectomy Sx 3%, due to Uveitis 2%.[5]

Sometimes the treatment can also lead to further complications, as the side effect of corticosteroid usage as well as the disease itself, patients with uveitis are more likely to have a rise in intraocular pressure.[6] Hence the treatment has to be carefully monitored.

Therapeutic modalities differ from one variety to another and what treatment is advised in one may not be the ideal treatment in the other type of sec.glaucoma.

In pigmentary dispersion syndrome which is a type of secondary open angle glaucoma, characterised by Trabecular Meshwork pigmentation, mid-peripheral radial iris transillumination abnormalities, and corneal endothelial pigment depositions on the posterior side of the cornea.[7]Lazer P.I. is considered a treatment modality and also has a protective role against rise of IOP over the coarse of 10yrs.[8]

Nd:YAG laser capsulotomy which is a method for treating posterior capsular opacification is known to disrupt the anterior hyaloid phase in 33% of instances, according to many publications.[9] It causes the anterior viterious to prolapse out of the posterior segment into the anterior segment and block the pupil leading to secondary glaucoma.

Therefore the medical management of secondary glaucoma is different from that of primary glaucoma as it requires additional treatment of the secondary cause & must be tailored to the patient. Surgical treatment of secondary glaucoma's also varies according to each type.

This hospital based study is to be done to bridge this gap of finding out the most common type of secondary glaucoma, the ratio of male and female, the mean age of people affected by secondary glaucoma and the various modalities of treating them.

Aims and Objectives

- Following is the aim of my study

  Prevention of blindness due to secondary glaucoma.

- Following are objectives

1. To study the various types of secondary glaucoma
2. To know the proportion of various types of secondary glaucoma
3. To evaluate the age wise distribution of various secondary glaucoma
4. To estimate the male:female ratio of secondary glaucoma
5. To study the various management of different types of secondary glaucoma

Review of literature

Glaucoma is a disease where the patient looses his/her sight irreversibly with intra-ocular pressure being the only modifiable factor. When a related or identifiable ophthalmic condition is known to be present with increase in IOP, it is referred to as secondary glaucoma. Patients usually present with diminished vision, headache, eye pain with VA ranging from 6/9 to HMCF. A few studies have been done to show the disease burden and the various treatments possible to prevent further damage to the retina, some are given below:

Lachovská Set al conducted a study of pseudo exfolation syndrome in which 14,167 patients were studied for PXS and 689 patients were diagnosed with it (6%) of cases had PXG with the study period being from November 2016 to April 2021.[10]

Talaat K et al at King Abdulaziz Medical City, Jeddah evaluated glaucoma in 2021 and concluded that secondary glaucoma constituted a total of 26.5% of all glaucoma patients and of those NVG was the most common 44.9% followed by lens induced glaucoma 29%.[11]

Gurung J et al in 2021 analyzed the profile of secondary glaucoma in a tertiary care hospital in Nepal and found that lens induced glaucoma was the most common 33%, followed by NVG 21%, proceeded by steroid induced glaucoma 16%, then came traumatic 15%.[5]

Gong H et al in 2021 researched on 11,730 cases with secondary glaucoma and found that the patients were 44.45 years old on average. Men were more susceptible (66.94%) than women (33.06%), the most common cause was trauma (28%), followed by vascular disease (18%), proceeded by uveitic (11%), then came lens induced (9%), then steroid induced (2%).[12]

Senthil S et al in 2021 conducted a study on 100 eyes with NVG and found that eyes that had NVG due to proliferative diabetic retinopathy had a higher rate of trab failure.[22]

Gao, F., Wang, J., Chen, J. et al (2021) studied angle closure glaucoma and found The second leading cause of angle closure glaucoma was uveitis (61 patients, 20.3%), then came NVG (4.9%), followed by ICE (7.2%)[13]

Komaratih E et al (2020) documented the profile of secondary glaucoma in Java and found that the male female ratio was 1.3:1 and the distribution of secondary glaucoma was lens induced 30.8% followed by steroid induced 29.5%, then came uveitic 20.5% and the least was NVG 15.4%.[14]

Krishnappa, Kanthamani et al.(2020) studied lens induced glaucoma and found that 23(47.9%) were males and 25(52.1%) were females, 32(66.7%) cases were of phacomorphic lens induced secondary glaucoma and 13 (27.1%) were of phacolytic and concluded that due to the delayed treatment of mature cataracts, LIG is a serious disease that can impair vision and affects the elderly.[15]

Dubey S et al. (2019) analyzed leading causes of secondary glaucoma. They were found to be NVG (17.42%), then came trauma (14.80%), followed by post-keratoplasty (13.60%), then post-cataract surgery (13.13%), and last was lens-induced glaucoma (12.41%).[16]

Senthil Set al in 2018 found that use of multifocal intraocular lens in presence of large capsulorrhexis should be avoided as they are prone for anterior displacement and can cause pigment dispersion glaucoma.[23]

Sherpa, D et al. (2017) Assessed that 35 (59.28%) were male and 24 (40.66%) were female secondary glaucoma patients, the male female ratio was 1.46:1, the most common secondary glaucoma was NVG with 52.54%,lens induced having 20.34%, then came uveitic 8.4%, then PXG 6.75%, followed by angle recession 3.39%, then pigmentary dispersion syndrome and pseudophakic glaucoma both came at 1.69%.[17]

Rifqah E et al.(2017) Conducted a study on secondary glaucoma in 63 patients and found that the most common age group affected was 40–59 years old (42.9%), 40 (63.5%) were men, 23 (36.5%) were females, the male : female ratio was 1.7:1 , the causes were lens induced (36.5%), followed by uveitic (22.2%), and the least was trauma (9.5%).[18]

Ajite KO et al. (2015) assessed the distribution of secondary glaucoma in Nigeria and found that post traumatic glaucoma 25(25.2%) was the most common, then came lens induced glaucoma 16 (16.2%), followed by steroid induced glaucoma 14 (14.2%), then came neovascular glaucoma 12 (12.1%), proceeded by uveitic glaucoma 10 (10.1%), followed by pseudoexfoliation glaucoma 10 (10.1%), the least was pigmentary glaucoma and aphakic glaucoma both having 6(6.1%).[19]

Yaakub A et al. (2014) researched on lens induced glaucoma and found that the mean age of presentation was 70.2 years and were predominantly women 22 (57.9%) affected, Phacomorphic glaucoma 28 (73.7%) was the main cause of LIG.[20]

Parivadhini A et al assessed various secondary angle closure glaucoma's management and found that all these diseases require careful and detailed history taking and meticulous examinations are required to prevent ocular morbidity.[21]

ripathy D et al found that thyroid eye disease leading to aqueous misdirection syndrome is a multi factorial cause like mechanical compression of trabecular meshwork, increase in episcleral venous pressure, increased pressure on the globe due to hypertrophy of the ocular muscles and required phcoemulsification with pars plana vitrectomy to manage the pressure.[24]

Krishnadas R et al studied secondary glaucoma and found that the treatment should depend whether the eyes have useful vision if so the goal of treatment should be to decrease IOP, lessen any accompanying inflammation, address the underlying cause (such as by removing a hyphaema or a hypermature lens), and do additional procedures to restore vision. Once the eye has become calm and stable, glaucoma surgery or long-term IOP control medication may be necessary.[25]

Coviltir V et al said ICE syndrome should be treated by Trabeculectomy but when it is ineffective in treating the disease the cause is usually extensive conjunctival scaring.[26]

Zemba M et al stated that UGH syndrome is a complication of cataract surgery, it causes secondary glaucoma and the treatment may necessitate IOL explantation.[27]

Lahola-Chomiak AA et al found that Retinal pigment epithelium also gets involved in pigmentary dispersion symdrome.[28]

Ritch R et al stated that eyes with pigment dispersion syndrome differ from normal eyes in that they have a larger iris, a midperipheral posterior iris concavity that grows with accommodation, a more posterior iris insertion, increased iridolenticular contact that is reversed by inhibition of blinking, and possibly an innate weakness of the iris pigment epithelium. Additionally, they have a higher incidence of lattice degeneration of the retina.[29]

Moraru A et al conducted a study on 38 eyes and found that the treatment of phacomorphic glaucoma, phacoemulsification and extracapsular extraction were both reliable and safe treatments that guaranteed a speedy functional recovery and good long-term IOP management. Despite the fact that individuals receiving phacoemulsification had a lower rate of acute postoperative problems and a better overall functional outcome.[30]

Materials and Methods

Organization: Department of ophthalmology, Santosh medical college and hospital , Ghaziabad

Study setting : OPD based

Study design: hospital based Clinical cross-sectional study

Study duration : 18 months

Study unit: 20 – 80 yrs.

Sample size: 105 cases was taken as sample size

Based on formula n =(Z1-α/2) 2P(1-P)/d2 where p=7.4% Prevalence (as per previous study)[5], z=1.96 at 5% significance, d= absolute error (5%) corresponding to effect size

n= 105.29=105

Patient selection: All secondary glaucoma Patient attending eye OPD at santosh hospital ghaziabad

Inclusion criteria: Patients aged 25-80 yrs medically diagnosed with secondary glaucoma

Exclusion criteria:

- All patients diagnosed with primary glaucoma's
- All patients diagnosed with developmental glaucoma's

Methodology

History : Detailed history was taken regarding the onset and progression of the ocular and systemic symptoms.

Visual Acuity : Using Snellen's digital chart visual acuity was determined from 6/6 to finger counting at 1m, projection of rays, perception of light.

Tonometry : The intraocular pressure was measured using Goldman applanation tonometer by making the patient sit on the slit lamp, staining with fluorescein Dye and installing a drop of Proparacain 0.5%. The prism was positioned on the cornea and applanated to form the mires, the reading is taken by rotating the dial. In some patients with corneal pathology a Non contact tonometer was used to measure the intraoccular pressure.

Slit Lamp Examination : The patient is made to sit on the slit lamp and examined in diffuse light for pupillary reaction, any corneal abnormality, iris details and lens opacities. On beam examination the corneal layers and endothelium, anterior chamber depth was measured using Van Herick method and other pathology cells, presence of any neovascularisation on iris, all are examined.

Gonioscopy : The patient was made to sit on the slit lamp, one drop of proparacain 0.5% was installed, using a coupling agent (HPMC) the 3 Mirror Gonioscope was placed on the cornea and using the 59° Mirror the angle of Anterior chamber was observed and is graded whether open or closed according to Shaffer's grading.

Slit lamp Bio microscopy : Using 78D Volk lens the patient was positioned on the slit lamp and the Optic disc was examined for the colour, size, shape, Cup**:**Disc, cup depth, any notching & Neuroretinal-rim.

Indirect ophthalmoscopy : It was performed using 20D Volk lens and an Appa indirect ophthalmoscope, the patient was dilated with tropicamide 0.5% drops for 3 times every 15min, the patient was made to lie in the dark room procedure bed and the fundus on a whole was examined for the fundal glow, presence of any Neo-vascularisation, venous or artereolar occlusion, chroidal patches & vitreous haemorrhage.

Management :

In cases of Phacomorphic Glaucoma: Intra venous manitol 300ml slowly with 2 Tab. Acetazolamide 250mg Stat was given to reduce the intra ocular pressure. After the pressure reduced and the patient was comfortable, the patient was shifted to the operation theater for undergoing emergency manual small incision cataract surgery and trabeculectomy combined procedure. The patient was followed up after 1,3,15,30 days

In cases of Neovascular Glaucoma: Intra venous Manitol 300ml slow with 2tabs of acetazolamide 250mg stat was given after which Intra-vitreal Ranibizumab 0.1ml was given, topical anti glaucoma drugs (timilol) was started twice daily and one week after pan retinal photo-coagulation was performed. The patient was followed up at 1,3,6 months.

In cases of Uveitic glaucoma : Topical Steroid (Prednisolone 1%) was given to decrease the inflammation 6-8times, topical cycloplegic & Mydriatic (Atropine1%) was given to decrease pain and to break synechae and a topical anti glaucoma drugs (timolol+Brimonidine combination) was given twice daily, in cases with pupillary block and iris bombay intravenous manitol 300ml slow with 2tabs of acetazolamide 250mg stat was given after which a Lazer Pheripheral Iridotomy was also performed. Patient was followed up after 1,2,3 weeks.

In cases of ICE (iridocorneal endothelial syndrome) **:** Topical anti glaucoma drugs (timilol) was started twice daily, trabeculectomy was performed and the patient was followed up after 1,2,3 weeks.

In cases of Pseudoexpholiation glaucoma : Oral & topical anti galucoma drugs (timolol + Brimonidine combination+ acetazolamide 250mg) are given twice daily and phacoemulsification is done to remove the lens. Patient is followed up for 6weeks.

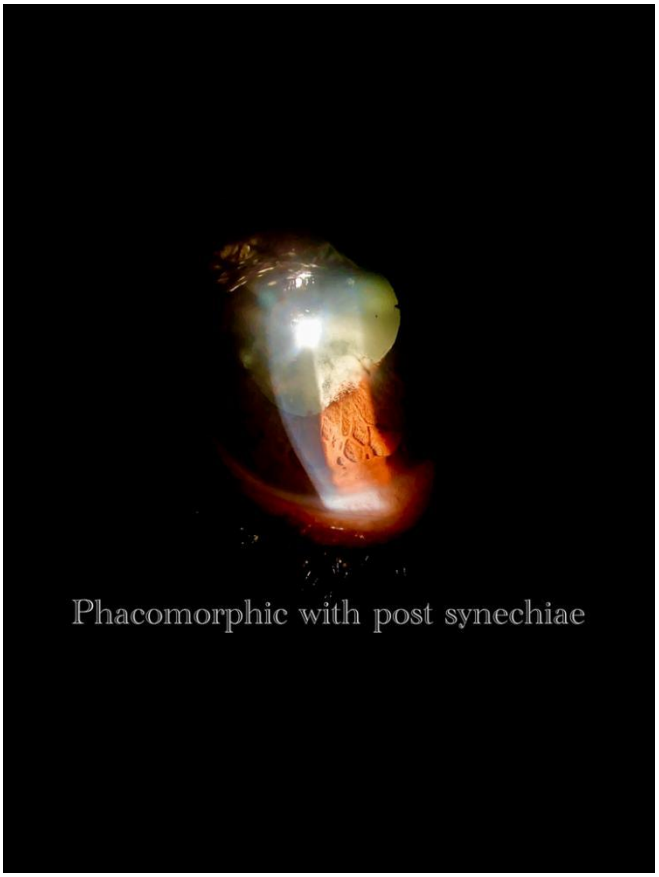
In cases of Traumatic Hyphema : If the Intra ocular pressure was less than 40mmhg, the patient received a topical mydriatic(Homatropine) + topical steroid (predforte) + topical anti glaucoma drugs(timolol + Brimonidine combination). If the pressure was greater than 40mmhg, the patient received iv. Manitol 300ml slow + tab. Acetazolamide 250mg X2 stat + topical anti glaucoma drugs TimololB + oral and topical steroid wysolone 20mg OD + Predforte 1% half hourly and the patient was taken up for AC wash. Patient is followed up daily in opd for 1week then weekly for 1 month.

In cases of steroid induced glaucoma : first the drug was stopped or if it couldn't be stopped the treating doctor was consulted and the patient was kept on the minimum dose. The patient was put on anti glaucoma drugs to acutely decrease the pressure. The patient was observed for 1 month if the iop still remained high then anti glaucoma drugs were continued.

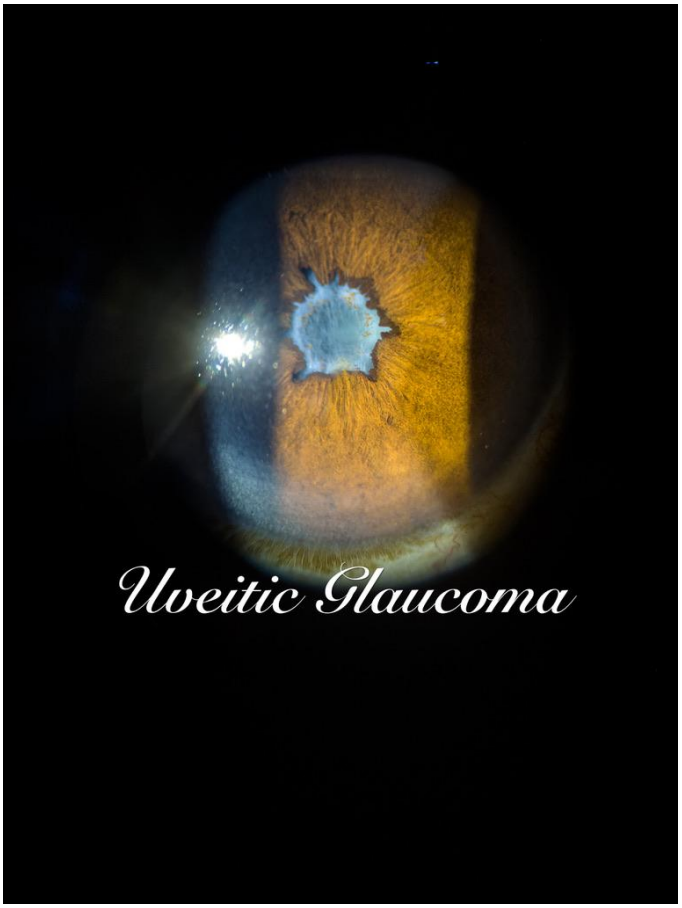
In pigmentary dispersion : Topical anti glaucoma drugs (timilol) was started twice daily and the patient was taken up for surgical peripheral iridotomy. The patient was fallowed up on 1,3,10,15 post op days.

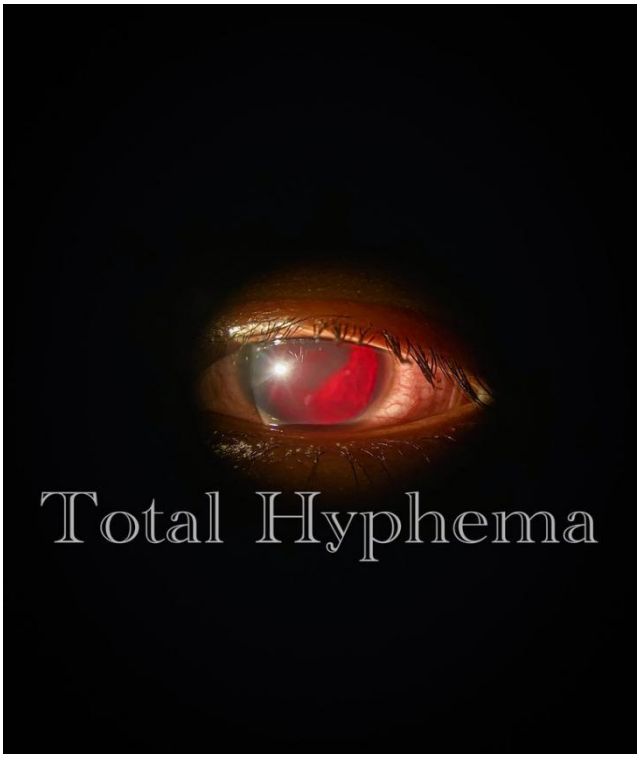
In silicone oil induced secondary glaucoma : The patient was put on topical anti glaucoma drugs and a glaucoma drainage device(Ahmed valve) was put in such patients.

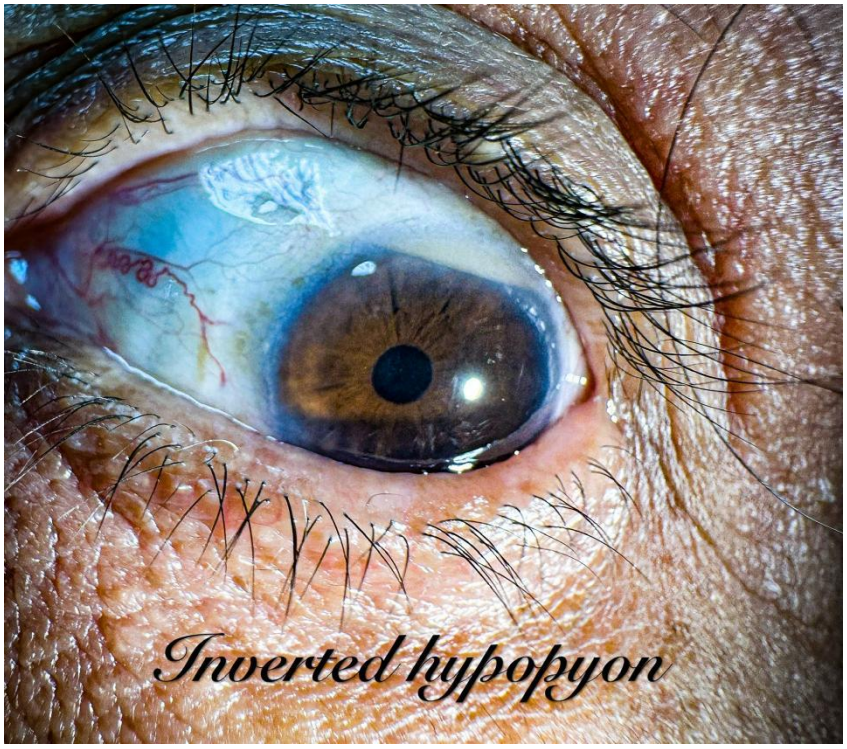
In patients of angle recession glaucoma : Oral anti-glaucoma drugs(acetazolamide 250mg), topical anti glaucoma drugs(timolol + Brimonidine combination)was given. The patient was taken up for Ahmed valve implantation later.

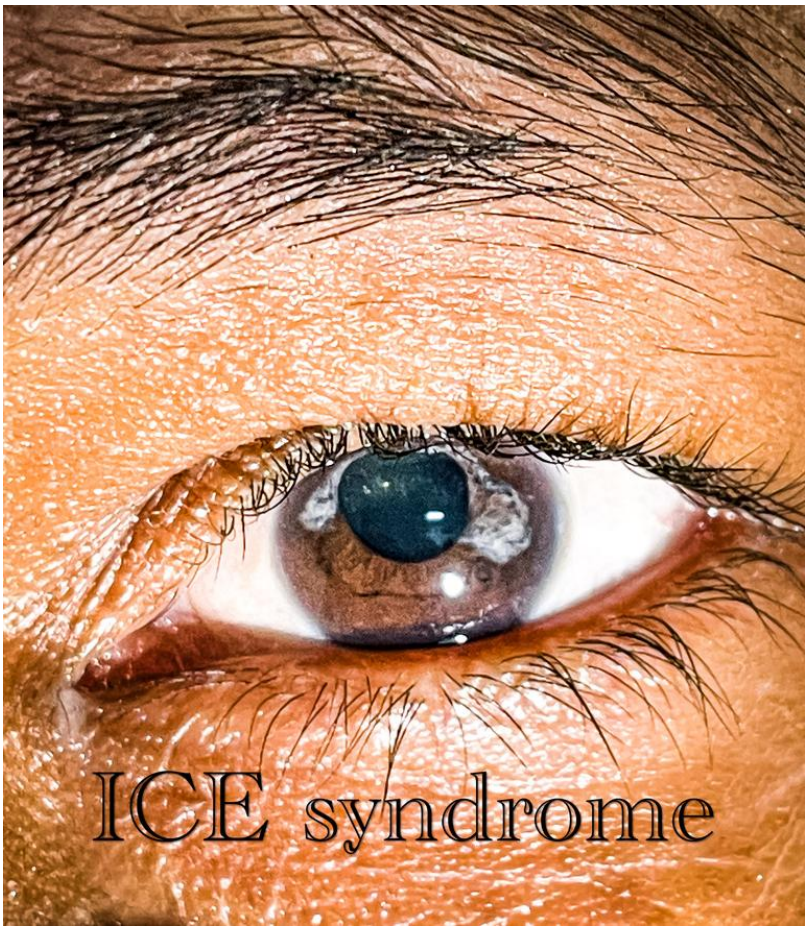

Phacomorphic with post synechiae

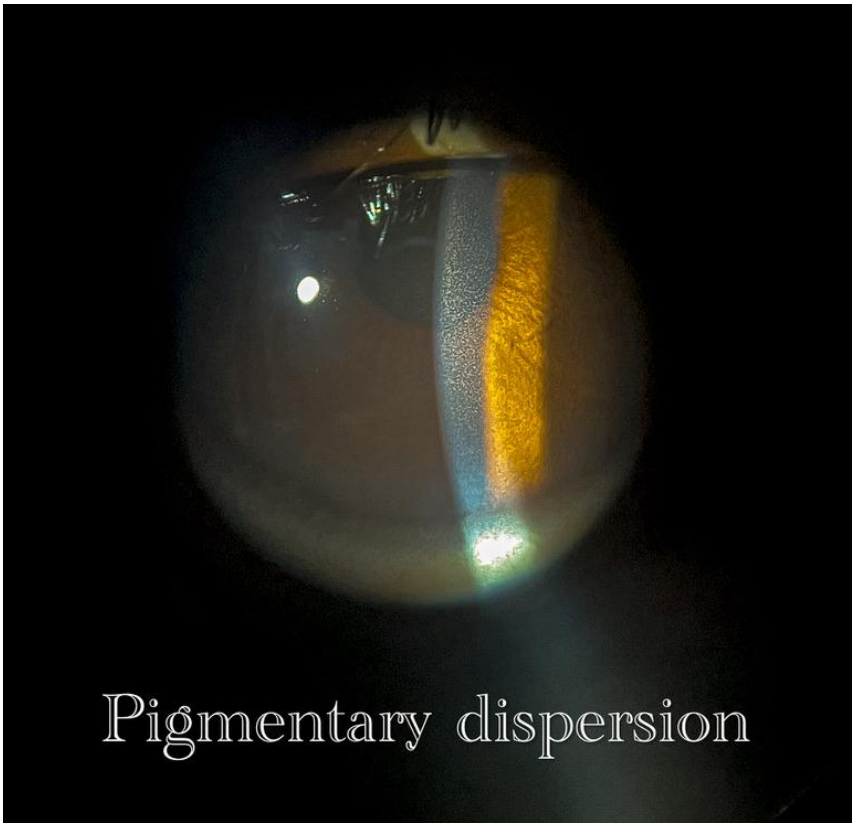
Neovascular Glaucoma

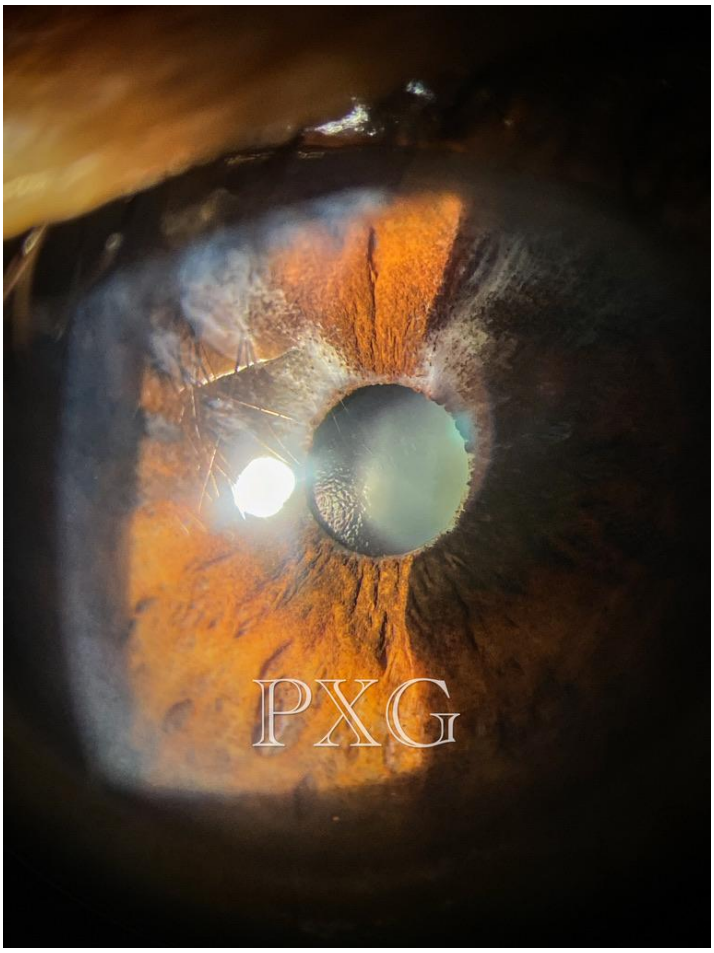

Uveitic Glaucoma


Total Hyphema

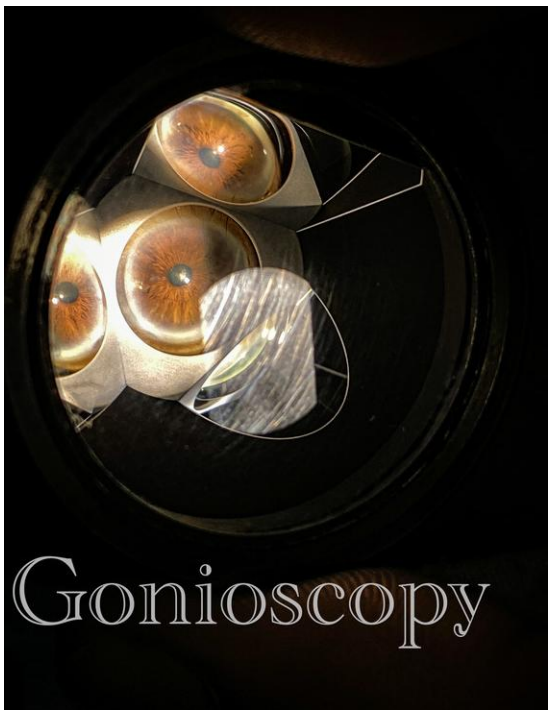

Inverted hypopyon

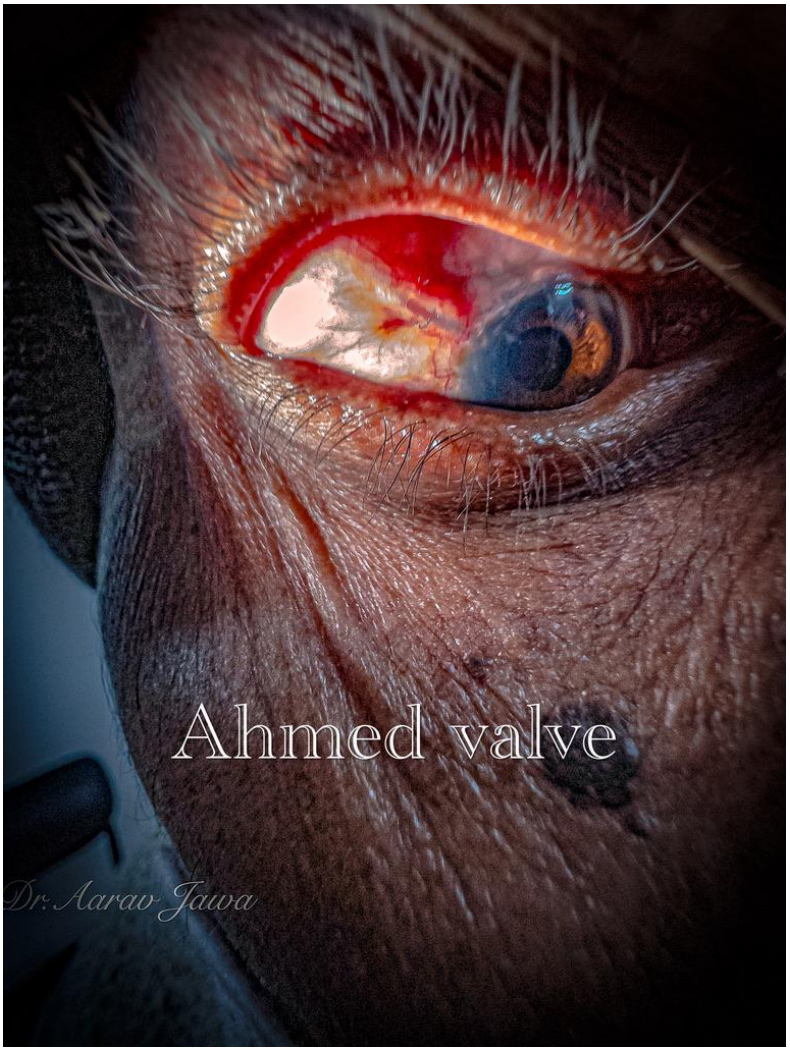

ICE syndrome


Pigmentary dispersion


PXG

78D examination


Gonioscopy


Ahmed valve
Dr. Aarav Jawa

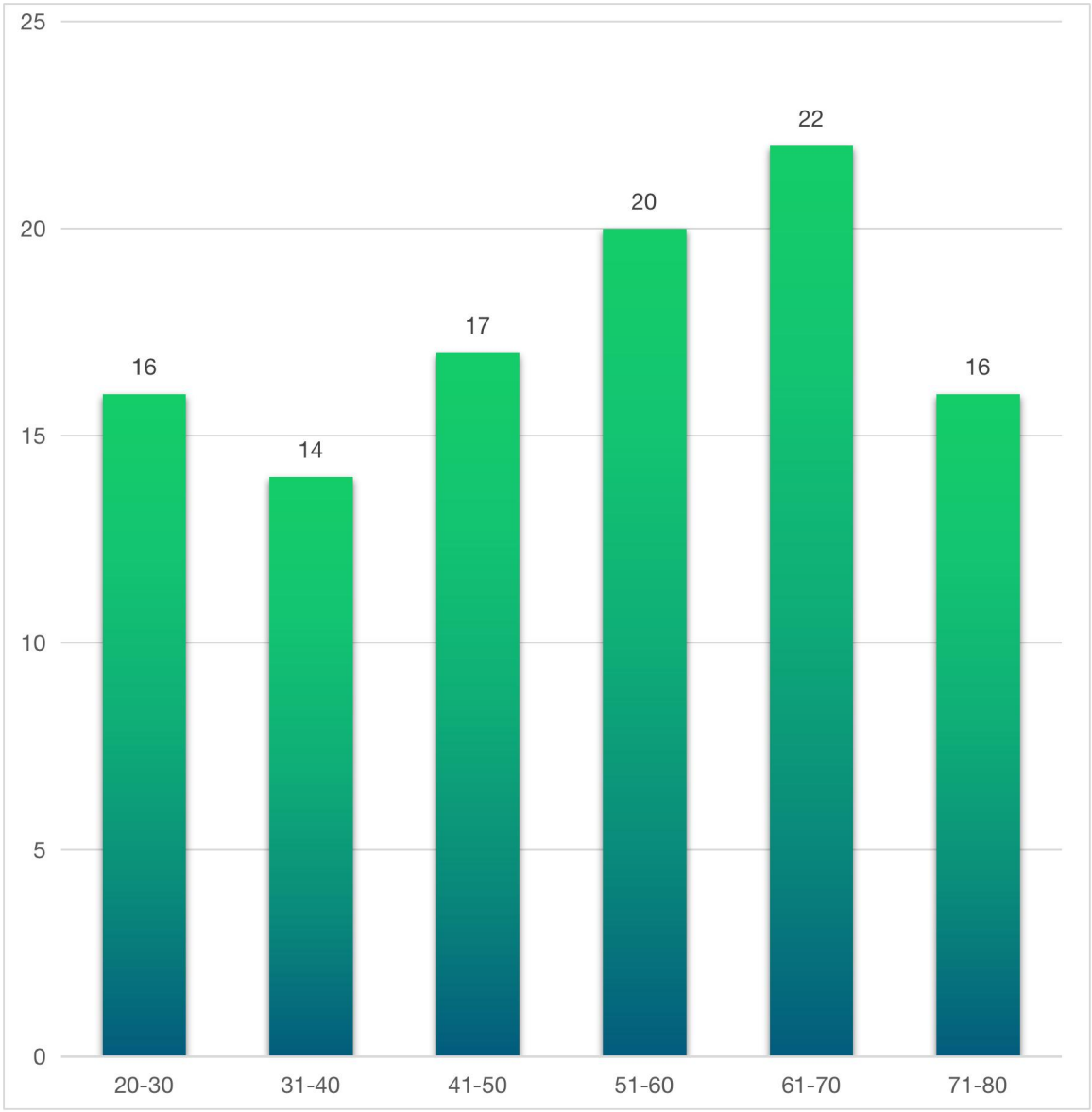
Results

A total of 105 cases of Secondary glaucoma were taken as part of this study. They included phacomorphic, neovascular, traumatic, uveitic, steroid induced, PXG, pigment dispersion, silicone oil induced, ICE, angle recession glaucoma.

Age distribution of secondary glaucoma		
Age	No. of patients	Percentage
20-30	16	15.2
31-40	14	13.2
41-50	17	16.2
51-60	20	19
61-70	22	21
71-80	16	15.2
Total	105	

Table1. Showing the age distribution of secondary glaucoma.

It was observed that the maximum number of patients belonged to the age group between 61-70yrs that is 22 patients (21%) followed by 51-60yrs having 20 patients (19%), 41-50yrs having 17 patients (16.2%), 71-80yrs & 20-30yrs having 16 patients each (15.2%) and 31-40 having the least amount of patients i.e. 14 (13.2%). This has been depicted in **Table1**

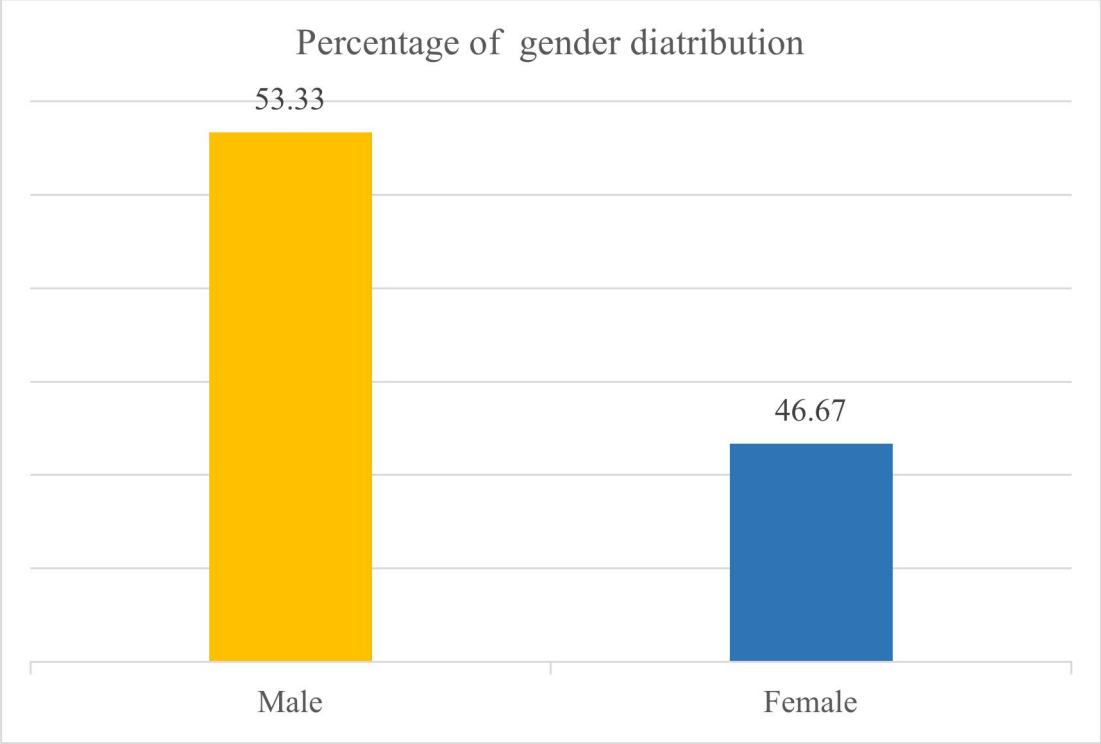


Graph 1. Depicting the age distribution of secondary glaucoma.

Gender	No. Of patients	Percent
Male	56	53.33
Female	49	46.67

Table.2 shows the gender distribution

Table.2 shows the gender distribution of secondary glaucoma.The males constituted 56 patients (53.33%), the females constituted 49 patients (46.7%). The male to female ratio was **1.4:1.** Males were 1.4 times more common to have secondary glaucoma.

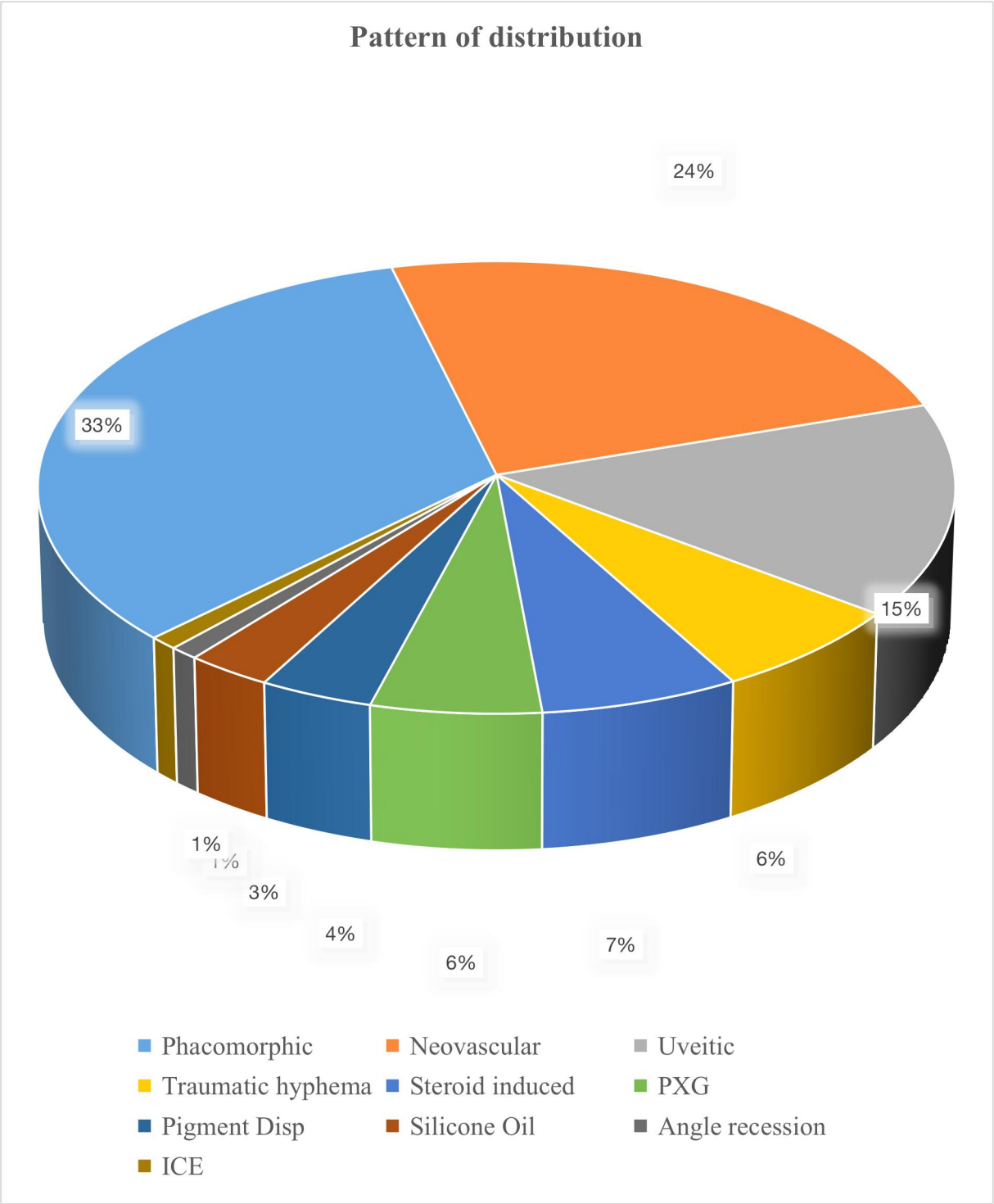


Graph.2 shows the gender distribution

Type of sec. glaucoma	No. Of cases	Percentage
Phacomorphic	35	33.3
Neovascular	25	23.8
Uveitic	16	15.3
Traumatic hyphema	7	6.6
Steroid induced	7	6.6
PXG	6	5.7
Pigment Disp	4	3.8
Silicone Oil	3	2.8
Angle recession	1	0.96
ICE	1	0.96
Total	105	100

Table.3 Various secondary glaucoma's encountered in this study.

Table.3 shows the verious secondary glaucoma's that were encountered during the study period. The most commonly encountered secondary glaucoma was Phacomorphic secondary angle closure glaucoma with a total of 35 cases (33.3%), proceeded by neovascular glaucoma with a total of 25 cases (23%), followed by uveitic glaucoma with 16 cases (15.3%), followed by traumatic hyphema and steroid induced glaucoma with 7 cases each (6.6%), then comes Pseudo exfolatiation glaucoma(PXG) with 6 cases (5.7%), then comes Pigment dispersion syndrome with a total of 4 cases (3.8%), proceeded by Silicone oil induced secondary glaucoma having 3 cases (2.8%), the least common were angle recession glaucoma and iridocorneal endothelial glaucoma(ICE) both having 1 case each (0.96%) out of 105 cases.

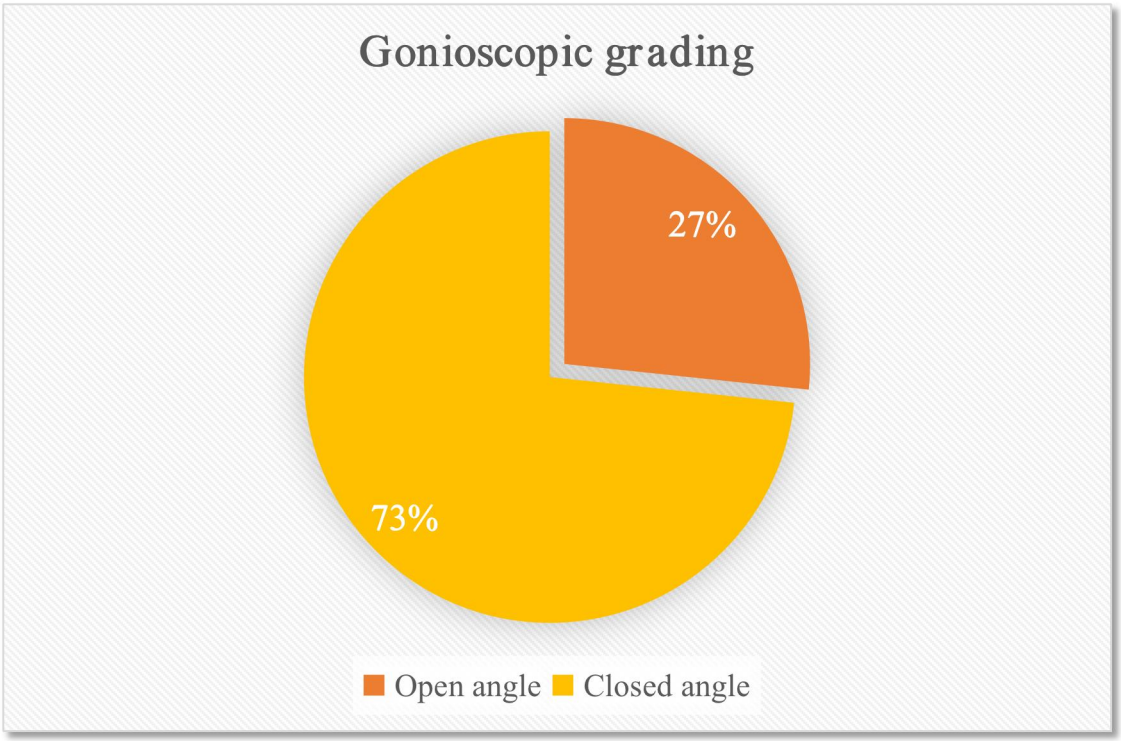


Graph.3 showing the distribution of various types secondary glaucoma

Gonioscopy	No. of patients	Percentage
Open angle	28	27
Closed angle	77	73

Table.4 shows the anterior chamber morphology

Table.4 shows us the anterior chamber morphology on gonioscopy. The patients with open angle secondary glaucoma were 28 (27%) whereas the patients with angle closure secondary glaucoma were 77 (73.%).

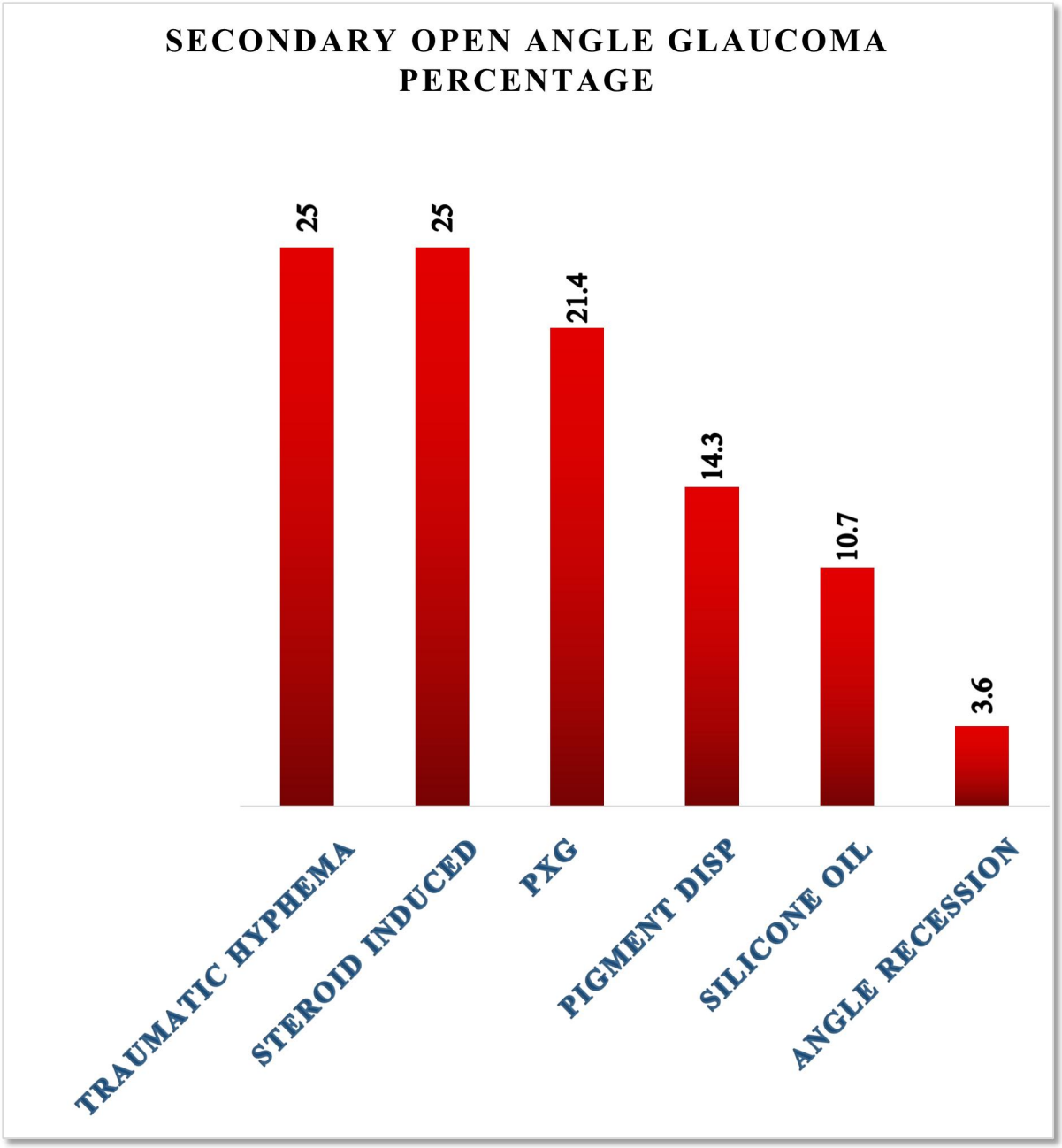


Graph.4 shows the percentage of open and closed angle patients acc to gonioscopy

Secondary Open angle		
Type	No. of patients	Percentage
Traumatic hyphema	7	25
Steroid induced	7	25
PXG	6	21.4
Pigment Disp	4	14.3
Silicone Oil	3	10.7
Angle recession	1	3.6
total	28	

Table.5 shows number of patients with different types of secondary open angle glaucoma.

Table.5 shows the number of secondary open angle glaucoma patients and its types that were encountred during this study. It consisted of a total of 28 patients out of 105 patients. The two most common secondary open angle glaucoma types encountered were traumatic hyphema and steroid induced both having 7 patients each (25%), followed by PXG having 6 patients (21.4%), then came pigment dispersion glaucoma with 4 cases (14.3%), preceded by silicone oil induced glaucoma with 3 cases and the least being angle recession with 1 case (3.6%). The total cases of secondary open angle glaucoma were 28.

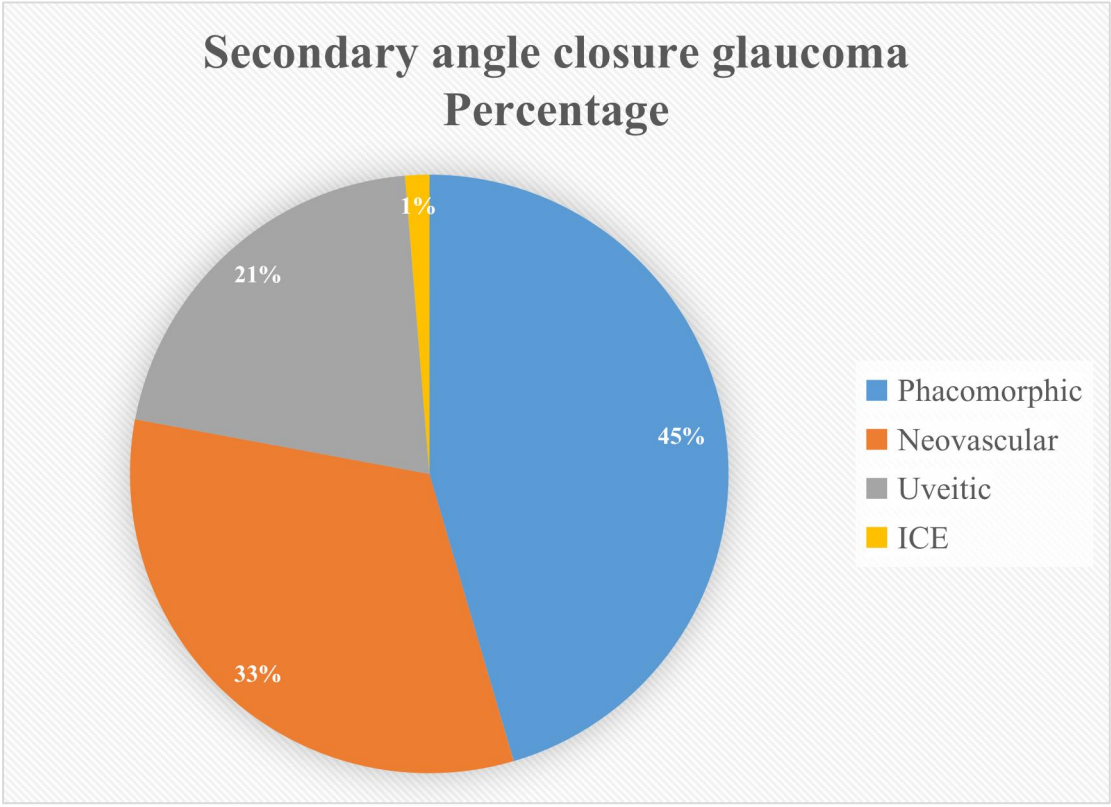


Graph.5 showing the distribution of various types of secondary open angle glaucoma.

Secondary angle closure glaucoma		
Type	No. of patients	Percentage
Phacomorphic	35	45.5
Neovascular	25	32.5
Uveitic	16	20.8
ICE	1	1.3
Total	77	

Table.6 shows the number of patients with different type of secondary angle closure glaucoma

Table.6 shows the various secondary angle closure glaucoma patients and their percentage. The most common type encountered was phacomorphic secondary angle closure glaucoma 35 cases (45.5%), then came neovascular secondary glaucoma with 25 cases (32.5%), preceded by uveitic secondary angle closure glaucoma with 16 cases (20.8%) and the least was ICE with 1 case (1.3%). The total cases of secondary angle closure glaucoma were 77



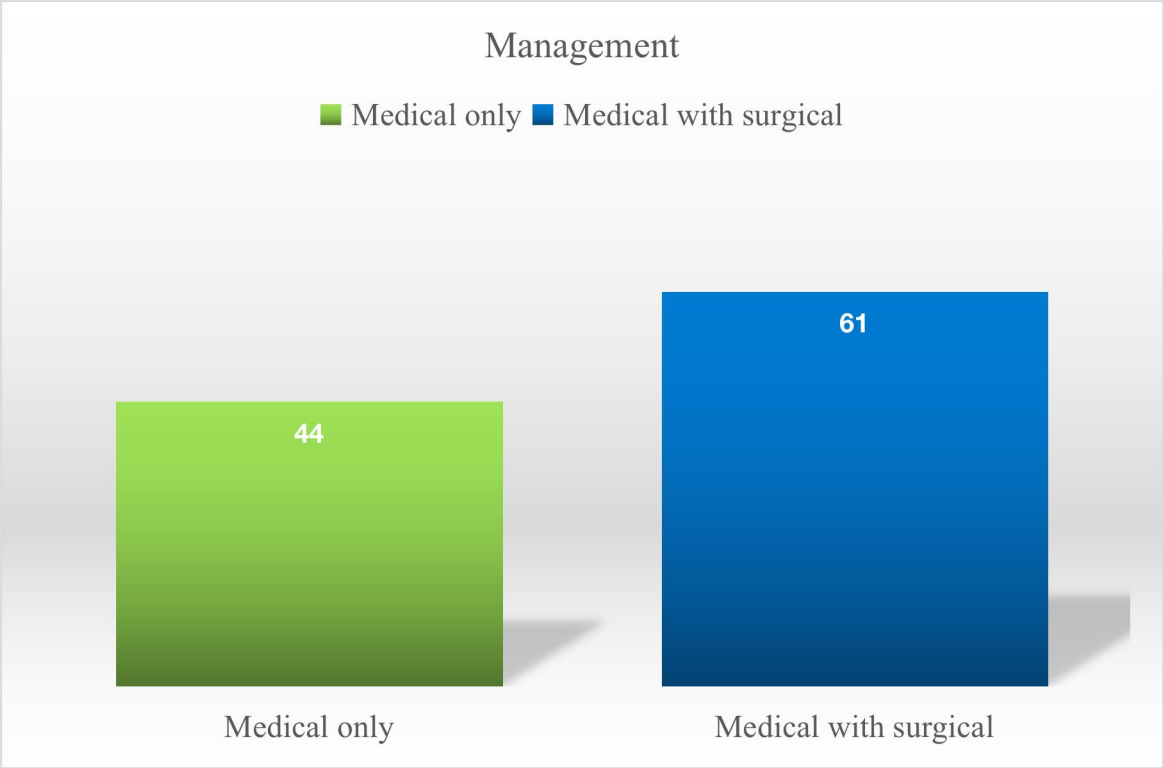
Graph.6 depicting the distribution of various types of secondary angle closure

glaucoma

Management	No. of patients	Percent
Medical only	44	41.90
Medical with surgical	61	58.10

Table.7 shows the management of secondary glaucoma

Table.7 shows the number and the percentage of patients that underwent either only medical management 44 patients (41.9%) or medical + surgical management 61 patients (58.10%).



Graph.7 shows the management

Discussion

WHO states that over 7.7 million persons with glaucomatous optic neuropathy are thought to exist in the globe.[2] It is estimated that there will be 111.8 million cases of glaucoma worldwide by 2040.[3] Studies regarding the current pattern of secondary glaucoma in our region haven't been performed.

This study included patients from the age of 20-80 yrs, with most patients of secondary glaucoma being in the age group of 61-70 that is 22 patients (21%) followed by 51-60yrs having 20 patients (19%), 41-50yrs having 17 patients (16.2%), 71-80yrs & 20-30yrs having 16 patients each (15.2%) and 31-40 having the least amount of patients i.e. 14 (13.2%). *Gong H et al* in 2021 also found that the mean age of the patients with secondary glaucoma was 44.45±19.45 years old.[12]

In this study the males constituted 56 patients (53.33%), the females constituted 49 patients (46.7%). The male to female ratio was **1.4:1.** Males were 1.4 times more common to have secondary glaucoma. *Sherpa, D et al* concluded that 35 (59.28%) were male and 24 (40.66%) were female with male and female ratio to be 1.46:1[17] and *Gong H et al* also found men (66.94%)[12] were more vulnerable than women to get secondary glaucoma.

In this study we found the patients with open angle secondary glaucoma were 28 (27%) whereas the patients with angle closure secondary glaucoma were 77 (73.%). The most common in open angle was traumatic hyphema and steroid induced both having 7 patients each (25%). The most common in angle closure was phacomorphic secondary angle closure glaucoma 35 cases (45.5%). *Ajite KO et al* also found traumatic hyphema to be the most common cause of secondary open angle glaucoma.[19]*Gurung J et al* also found the most common cause of secondary angle closure glaucoma was lens induced glaucoma.[5]

We found that most patients required medical plus surgical treatment for long term control of intra-ocular pressure (58.10%). *Krishnadas R et al* said that the treatment depends on weather the patients has useful vision if so the goal of treatment should be to decrease IOP, lessen any accompanying inflammation, address the underlying cause (such as by removing a hyphaema or a hypermature lens), and do additional procedures to restore vision. Once the eye has become calm and stable, glaucoma surgery or long-term IOP control medication may be necessary.[25]

We found that PXG can be treated and controlled by Oral & topical anti galucoma drugs (timolol + Brimonidine combination+ acetazolamide 250mg) are given twice daily and phacoemulsification while *Hemalatha BC* et al conducted MSICS in most cases[31]

We found phacomorphic glaucoma to be treated by Intra venous manitol 300ml slowly with 2 Tab. Acetazolamide 250mg Stat, manual small incision cataract surgery and trabeculectomy combined procedure. *Moraru A et al* conducted a study on 38 eyes and found that the treatment with phacoemulsification or extracapsular extraction was enough to control IOP.[30]

Summary

A total of 105 cases of Secondary glaucoma were taken as part of this study. They included phacomorphic, neovascular, traumatic, uveitic, steroid induced, PXG, pigment dispersion, silicone oil induced, ICE, angle recession glaucoma.

The gender distribution of secondary glaucoma in this study was Males 53.33% and females 46.7%. The male female ratio was **1.4:1.**

Phacomorphic secondary angle closure glaucoma was the most common with a total of 35 cases (33.3%), proceeded by neovascular glaucoma with a total of 25 cases (23%).The least common were angle recession glaucoma and iridocorneal endothelial glaucoma(ICE) both having 1 case each (0.96%)

58.1% patients required surgical intervention where as 41.9% patients were controlled with medical management only.

Conclusion

I would like to conclude that secondary glaucoma is very common in the older age and low socioeconomic group as they tend to prolong the treatment for cataract and in the end lead to secondary angle closure glaucoma, people with diabetes & hypertension should get regular ocular checkups including gonioscopy as they have a tendency for neovascularisation. Patients on steroids oral and topical should be monitored and screened for secondary steroid induced glaucoma hence the medical practitioner treating such patients should be made aware regarding the repercussions. Patients with TB, rheumatoid artheritis, hyperlipidemia, should be monitored closely as they have the tendency to develop secondary uveitic glaucoma. In this study we also found that secondary angle closure glaucoma than secondary open angle glaucoma, most patients required some form surgical intervention.

Bibliography

1. Davis BM, Crawley L, Pahlitzsch M, Javaid F, Cordeiro MF (December 2016). "Glaucoma: the retina and beyond". *Acta Neuropathologica*. 132 (6): 807–826. doi:10.1007/s00401-016-1609-2. PMC 5106492. PMID 27544758

2. GBD 2019 Blindness and Vision Impairment Collaborators; Vision Loss Expert Group of the Global Burden of Disease Study. Causes of blindness and vision impairment in 2020 and trends over 30 years, and prevalence of avoidable blindness in relation to VISION 2020: the Right to Sight: an analysis for the Global Burden of Disease Study. Lancet Glob Health. 2021 Feb;9(2):e144-e160. doi: 10.1016/S2214-109X(20)30489-7.

3. Tham YC, Li X, Wong TY, Quigley HA, Aung T, Cheng CY. Global prevalence of glaucoma and projections of glaucoma burden through 2040: a systematic review and meta-analysis. Ophthalmology. 2014 Nov;121(11):2081-90. doi: 10.1016/j.ophtha.2014.05.013. Epub 2014 Jun 26. PMID: 24974815.

4. Ahmad SZ, Ahmad A, Khalique N, Alvi Y. A community based study of scenario of glaucoma in Aligarh, India. Int J Community Med Public Health 2019;6:4098-104.

5. Gurung J, Sitoula RP, Singh AK. Profile of Secondary Glaucoma in a Tertiary Eye Hospital of Eastern Nepal. Nepal J Ophthalmol. 2021 Jan;13(25):98-103. doi: 10.3126/nepjoph.v13i1.28968. PMID: 33981103.

6. Siddique S. S., Suelves A. M., Baheti U., Foster C. S. Glaucoma and uveitis. Survey of Ophthalmology. 2013;58(1):1–10.doi: 10.1016/j.survophthal.2012.04.006

7. SUGAR HS, BARBOUR FA. Pigmentary glaucoma; a rare clinical entity. Am J Ophthalmol. 1949 Jan;32(1):90-2

8. Gandolfi SA, Ungaro N, Tardini MG, Ghirardini S, Carta A, Mora P. A 10-year follow-up to determine the effect of YAG laser iridotomy on the natural history of pigment dispersion syndrome: a randomized clinical trial. JAMA Ophthalmol. 2014 Dec;132(12):1433-8

9. Li EY-mei, Wu W-K, Jhanji V. Pupillary block glaucoma secondary to vitreous prolapse after Nd:YAG capsulotomy. *Clin Exp Optom* 2011;94:383–4. 10.1111/j.1444-0938.2010.00577.

10. Lachovská S, Kristian P, Krowicka. INCIDENCE OF PSEUDOEXFOLIATION SYNDROME AND GLAUCOMA IN A SET OF MORE THAN 14,000 EYES OF PATIENTS OPERATED FOR A CATARACT. Cesk Slov Oftalmol. 2022 Spring;78(2):56-62. English. doi: 10.31348/2022/8. PMID: 35477245.

11. Talaat K, Fathi OT, Alamoudi SM, Alzahrani MG, Mukhtar RM, Khan MA. Types of Glaucoma and Associated Comorbidities Among Patients at King Abdulaziz Medical City, Jeddah. Cureus. 2021 Jun 10;13(6):e15574. doi: 10.7759/cureus.15574. PMID: 34277196; PMCID: PMC8270073.

12. Gong H, Ren J, Zheng B, Huang X, Liao Y, Zhou Y, Lin M. The Profile of Secondary Glaucoma in China: A Study of Over 10,000 Patients. J Glaucoma. 2021 Oct 1;30(10):895-901. doi: 10.1097/IJG.0000000000001924. PMID: 34366389.

13. *Gao, F., Wang, J., Chen, J. et al. Etiologies and clinical characteristics of young patients with angle-closure glaucoma: a 15-year single-center retrospective study. Graefes Arch Clin Exp Ophthalmol 259, 2379–2387 (2021). https://doi.org/10.1007/s00417-021-05172-6*

14. Komaratih E, Rindiastuti Y, Primitasari Y. Profile of Secondary Glaucoma at a Tertiary Hospital in East Java. Folia Medica Indonesiana. 2020;56(1):56-9.

15. *Krishnappa, Kanthamani et al. "Clinical profile, visual outcome and complications after cataract surgery in patients with lens induced glaucoma." Indian Journal of Clinical and Experimental Ophthalmology (2020): n. pag.*

16. Dubey S, Jain K, Mukherjee S, Sharma N, Pegu J, Gandhi M, Bhoot M. Current profile of secondary glaucoma in a Northern India tertiary eye care hospital. Ophthalmic Epidemiol. 2019 Jun;26(3):200-207. doi: 10.1080/09286586.2019.1574840. Epub 2019 Feb 4. PMID: 30714467

17. *Sherpa, D. and Pokhrel, S. (2017) "Current pattern of secondary glaucoma", Journal of Chitwan Medical College, 7(3), pp. 21–24. Available at: https://www.nepjol.info/index.php/JCMC/article/view/23690 (Accessed: 7 November 2022)*

18. Rifqah E, Gustianty E, Prajitno IP. One year data of new secondary glaucoma patients at top referral eye hospital in Indonesia. Althea medical journal. 2017 Jun 30;4(2):163-6.

19. Ajite KO, Fadamiro CO, Ajayi IA, Omotoye OJ. Pro-file of Secondary Glaucoma in a Semi Urban Tertiary Health Care Hos-pital In Nigeria. Int J Ophthalmol Eye Res. 2015 Mar 25;3(3):97-100.

20. Yaakub A, Abdullah N, Siti Raihan I, Ahmad Tajudin LS. Lens-induced glaucoma in a tertiary centre in northeast of Malaysia. Malays Fam Physician. 2014 Aug 31;9(2):48-52. PMID: 25883765; PMCID: PMC4391518.

21. Parivadhini A, Lingam V. Management of Secondary Angle Closure Glaucoma. J Curr Glaucoma Pract. 2014 Jan-Apr;8(1):25-32. doi: 10.5005/jp-journals-10008-1157. Epub 2014 Jan 16. PMID: 26997804; PMCID: PMC4741163.

22. Senthil S, Chary R, Ali MH, Cherukuri JR, Rani PK, Krishnamurthy R, Choudhari N, Garudadri C. Trabeculectomy for neovascular glaucoma in proliferative diabetic retinopathy, central retinal vein occlusion, and ocular ischemic syndrome: Surgical outcomes and prognostic factors for failure. Indian J Ophthalmol. 2021 Nov;69(11):3341-3348. doi: 10.4103/ijo.IJO_1516_21. PMID: 34708802; PMCID: PMC8725114.

23. Senthil S, Grover IG. In-the-bag multifocal intraocular lens causing pigment dispersion and refractory secondary ocular hypertension. Indian J Ophthalmol. 2018 Sep;66(9):1339-1341. doi: 10.4103/ijo.IJO_198_18. PMID: 30127166; PMCID: PMC6113817.

24. ripathy D, Rao A, Banerjee A, Padhy D. Aqueous misdirection in thyroid eye disease. BMJ Case Rep. 2014 Nov 20;2014:bcr2014205824. doi: 10.1136/bcr-2014-205824. PMID: 25414214; PMCID: PMC4244329.

25. Krishnadas R, Ramakrishnan R. Secondary glaucomas: the tasks ahead. Community Eye Health. 2001;14(39):40-2. PMID: 17491925; PMCID: PMC1705934.

26. Coviltir V, Valentin D. ICE SYNDROME--CASE REPORT. Rom J Ophthalmol. 2015 Apr-Jun;59(2):119-22. PMID: 26978875; PMCID: PMC5712930.

27. Zemba M, Camburu G. Uveitis-Glaucoma-Hyphaema Syndrome. General review. Rom J Ophthalmol. 2017 Jan-Mar;61(1):11-17. doi: 10.22336/rjo.2017.3. PMID: 29450365; PMCID: PMC5710046.

28. Lahola-Chomiak AA, Walter MA. Molecular Genetics of Pigment Dispersion Syndrome and Pigmentary Glaucoma: New Insights into Mechanisms. J Ophthalmol. 2018 Mar 26;2018:5926906. doi: 10.1155/2018/5926906. PMID: 29780638; PMCID: PMC5892222.

29. Ritch R. A unification hypothesis of pigment dispersion syndrome. Trans Am Ophthalmol Soc. 1996;94:381-405; discussion 405-9. PMID: 8981706; PMCID: PMC1312105.

30. Moraru A, Pînzaru G, Moţoc A, Costin D. Functional results of cataract surgery in the treatment of phacomorphic glaucoma. Rom J Ophthalmol. 2017 Jul-Sep;61(3):202-206. doi: 10.22336/rjo.2017.37. PMID: 29450399; PMCID: PMC5710039.

31. Hemalatha BC, Shetty SB. Analysis of Intraoperative and Postoperative Complications in Pseudoexfoliation Eyes Undergoing Cataract Surgery. J Clin Diagn Res. 2016 Apr;10(4):NC05-8. doi: 10.7860/JCDR/2016/17548.7545. Epub 2016 Apr 1. PMID: 27190848; PMCID: PMC4866146.

Annexures

Proforma

1. Patient particulars
   a) Name:
   b) Age:
   c) Sex:
   d) Occupation:
   e) Address:
2. History
   a) Symptoms
   b) Duration
   c) Associated complaints
   d) Ocular Trauma
   e) Ocular Surgery
   f) Medications
   g) Systemic illness
3. General physical examination
4. Oculaar examination
   a) Visual acuity
   b) Gaze
   c) Lids & adenexa
   d) Conjunctiva
   e) Sclera
   f) Cornea
   g) AC
   h) Iris
   i) Pupil
   j) Lens
   k) Iop
   l) Gonioscopy
   m) Fundus

5. Investigations
    a) Blood sugar level
    b) Blood pressure
    c) Visual fields
    d) B-scan
    e) Chest x ray
6. Management
    a) Medical: Topical, intravenous IOP lowering drugs
    b) Surgical: Manual SICS, LASER Pheripheral Iridotomy, surgical Pheripheral Iridotomy, Trabeculectomy, AC wash.
7. Follow up
    a) IOP
    b) Visual acuity
    c) Fundus
    d) Gonioscopy

KEY TO MASTER CHART

PRN : OPD number

Rx : Treatment

Gonio : Gonioscopy

VA : Visual acuity

F : Female

M : Male

Lazer PI : lazer Pheripheral Iridotomy

Surgical PI : surgical Pheripheral Iridotomy

Trab : Trabeculectomy

MSICS : Manual small incision cataract surgery

AC : Anterior chamber

Phaco : Phacoemulsification

HMCF : Hand movements close to face

PL : Perception of light

IV Rani : Intravitreal Ranibizumab

Lazer : Panretinal photocoagulation

Ahm valve : Ahmed valve

Pigment Disp : Pigment dispersion glaucoma

ICE : Iridocorneal Endothelial Syndrome

PXG : Pseudo exfoliation glaucoma

Master Chart											
Sr.No.	PRN	Age	Sex	Intra ocular pressure		Gonio	Diagnosis	Rx		VA	
				Before Rx	After Rx			Medical	Surgical	Before RX	After Rx
1	17084418	60	F	56	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/12
2	17084697	60	F	50	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/12
3	17083795	52	M	56	17	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	1/60	6/9
4	17090461	48	F	60	14	Closed	Uveitic	Manitol + Acetazolamide + TimololB + Atropine + Predforte	lazer PI	6/24	6/12
5	17091584	20	M	52	16	Open	Traumatic hyphema	Manitol + Acetazolamide + TimololB + wysolone + Predforte	Ac wash	PL+	6/12
6	17091373	45	M	28	15	Closed	Uveitic	Homide + Predforte + TimololB		6/36	6/9
7	17092256	65	F	55	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/12
8	15002391	70	F	60	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
9	17092213	40	M	60	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
10	17098466	60	F	30	18	Open	PXG	Acetazolamide + Combigan	Phaco	6/24	6/9
11	17128231	70	M	48	17	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/24
12	17096564	60	F	25	15	Closed	Angle recession	Acetazolamide + Combigan	Ahm valve	6/9	6/9
13	17101697	31	F	42	18	Closed	Uveitic	Manitol + Acetazolamide + TimololB + Atropine + Predforte	lazer PI	6/24	6/9
14	17107710	45	F	48	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
15	17107508	44	F	40	18	Open	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/12
16	17108103	50	M	36	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/24
17	17103807	75	F	52	17	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/12
18	17128231	70	F	45	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/12
19	17185780	50	M	38	18	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/6
20	17192718	26	M	30	16	Closed	Uveitic	Homide + Predforte + TimololB		6/36	6/6
21	17192719	45	M	62	19	Open	Traumatic hyphema	Manitol + Acetazolamide + TimololB + wysolone + Predforte	Ac wash	PL+	6/12
22	17192720	40	M	45	22	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/24	6/9
23	17192747	46	M	24	18	Open	Pigment Disp	TimololB	Surgical PI	6/24	6/6
24	17192750	30	M	36	18	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/12
25	17192799	50	M	48	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/12
26	17192839	25	M	28	20	Open	Traumatic hyphema	Homide + Predforte + TimololB		HMCF	6/6
27	17195279	30	M	34	20	Open	Traumatic hyphema	Homide + Predforte + TimololB		HMCF	6/9

28	17194703	30	F	28	19	Closed	Uveitic	Homide + Predforte + TimololB		6/24	6/9
29	17196986	55	F	38	20	Closed	Uveitic	Manitol + Acetazolamide + TimololB + Atropine + Predforte	lazer PI	6/36	6/9
30	17199254	63	F	28	18	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/12
31	17201393	22	F	26	18	Closed	Uveitic	Homide + Predforte + TimololB		6/36	6/6
32	17202674	68	F	40	20	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
33	17209146	55	F	43	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
34	17205135	30	M	60	24	Closed	Uveitic	Manitol + Acetazolamide + TimololB + Atropine + Predforte	lazer PI	6/24	6/9
35	17206435	28	M	35	18	Open	Steroid induced	TimololB		6/9	6/6
36	17206816	46	M	42	19	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/9
37	17206850	60	M	56	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
38	17206864	35	F	38	16	Closed	Uveitic	Homide + Predforte + TimololB		6/24	6/9
39	17206840	69	F	46	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
40	17206848	30	M	26	17	Closed	ICE	Acetazolamide + Combigan	Trab	6/24	6/9
41	17206888	60	F	48	14	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
42	17206828	80	F	60	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
43	17206839	36	M	48	15	Closed	Uveitic	Manitol + Acetazolamide + TimololB + Atropine + Predforte	lazer PI	6/60	6/9
44	17206884	65	M	60	16	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/24
45	17206895	70	F	58	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
46	17206913	40	M	49	16	Closed	Uveitic	Manitol + Acetazolamide + TimololB + Atropine + Predforte	lazer PI	6/60	6/9
47	17206970	75	M	56	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
48	17207024	29	M	30	17	Open	Steroid induced	TimololB		6/9	6/6
49	17207098	50	F	40	15	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/9
50	17207346	30	M	30	18	Open	Steroid induced	TimololB		6/24	6/6
51	17207398	70	F	55	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
52	17207440	55	M	46	18	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/9
53	17207456	40	M	36	20	Closed	Uveitic	Homide + Predforte + TimololB		6/24	6/6
54	17209463	30	F	30	17	Open	Steroid induced	TimololB		6/9	6/6
55	2206020396	70	F	55	20	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
56	2206020506	35	M	35	18	Closed	Uveitic	Homide + Predforte + TimololB		6/24	6/6
57	2206020954	30	F	29	16	Open	Steroid induced	TimololB		6/12	6/6

58	2206021000	79	M	58	19	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
59	2206021494	36	M	30	22	Open	Steroid induced	TimololB		6/12	6/6
60	2206021896	76	F	60	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
61	17210136	78	M	46	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
62	17210286	80	F	48	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/12
63	17210403	48	F	46	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/24
64	17210600	40	M	48	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/24
65	17210765	68	M	60	19	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
66	17210796	50	M	50	20	Open	Silicone Oil	TimololB	Ahm valve	6/60	6/60
67	17210856	75	M	56	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
68	17210912	60	M	50	18	Open	Silicone Oil	TimololB	Ahm valve	6/60	6/60
69	17210981	67	F	56	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/24	6/12
70	17211245	78	F	60	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
71	17211459	56	M	30	18	Open	PXG	Acetazolamide + Combigan	Phaco	6/36	6/9
72	17211581	46	M	32	17	Closed	Uveitic	Homide + Predforte + TimololB		6/60	6/12
73	17211655	59	F	45	14	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/12
74	17214221	40	F	54	16	Open	Pigment Disp	TimololB	Surgical PI	6/12	6/6
75	17216730	72	M	60	15	Open	Traumatic hyphema	Manitol + Acetazolamide + TimololB + wysolone + Predforte		PL+	6/9
76	17216798	56	F	58	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/6
77	17216857	62	M	40	18	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/24	6/9
78	17216943	74	M	40	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
79	17207635	46	M	45	18	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/9
80	17202563	40	F	28	17	Open	Pigment Disp	TimololB	Surgical PI	6/24	6/9
81	17202754	63	F	58	15	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/12
82	17219463	76	M	55	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
83	17219629	65	M	32	16	Open	PXG	Acetazolamide + Combigan	Phaco	6/36	6/12
84	17222151	42	F	45	18	Open	Pigment Disp	TimololB	Surgical PI	6/12	6/6
85	17208936	53	M	40	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/12
86	17220136	79	M	50	17	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/12
87	17221265	55	M	38	20	Closed	Uveitic	Manitol + Acetazolamide + TimololB + Atropine + Predforte	lazer PI	6/36	6/9

88	17223469	63	F	60	18	Open	Traumatic hyphema	Manitol + Acetazolamide + TimololB + wysolone + Predforte	Ac wash	PL+	6/6
89	17224726	66	F	56	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
90	17224953	28	F	48	19	Open	Traumatic hyphema	Manitol + Acetazolamide + TimololB + wysolone + Predforte	Ac wash	PL+	6/9
91	17225230	57	M	40	22	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/12	6/6
92	17225610	34	M	30	18	Closed	Uveitic	Homide + Predforte + TimololB		6/36	6/6
93	17226159	57	F	30	18	Open	PXG	Acetazolamide + Combigan	Phaco	6/36	6/9
94	17226859	77	M	50	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
95	17227035	62	F	32	20	Open	PXG	Acetazolamide + Combigan	Phaco	6/60	6/9
96	17227269	26	M	45	20	Open	Steroid induced	TimololB		6/24	6/6
97	17227631	56	F	40	19	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/36	6/9
98	17227916	49	F	50	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/9
99	17228269	63	M	28	18	Open	PXG	Acetazolamide + Combigan	Phaco	6/24	6/6
100	17228634	80	M	55	18	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9
101	17228942	59	F	48	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/9
102	17229597	67	M	30	19	Open	Silicone Oil	TimololB	Ahm valve	6/60	6/60
103	17229842	75	F	56	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/12
104	17230248	40	M	26	20	Closed	Neovascular	IV Rani+ lazer+ Manitol+ Acetazolamide+Tomolol		6/60	6/9
105	17230384	65	F	50	16	Closed	Phacomorphic	Manitol + Acetazolamide	MSICS + trab	HMCF	6/9

Consent

DR. Aarav Jawa has explained about the risk and procedure. I volunteer to take part in the study entitled “Clinical study and management of secondary glaucoma” "and have also been explained the purpose of the study to my satisfaction.You are being requested to take part in a study entitled - Clinical study and management of secondary glaucoma

VOLUNTARY PARTICIPATIONS

Your participations in this study is entirely voluntary and you are free to withdraw from this study as and you feel so.

RISK (IF ANY)

Adequate care will be taken if patient faces any adverse effect with the drug being used during study.

FORM

I.....S/o/D/o.....

.....

R/o..... has been explained by

I hereby give my consent to be enrolled in the study.

Santosh Medical College and Hospital

Informed Consent

I. .....S/D/W of.....

Resident of.....

Hereby declare that I give my full and free & voluntary informed consent in the thesis entitled **"Clinical study and management of secondary glaucoma"**

DR.Aarav Jawa has informed to me to my full satisfaction, in the language I understand about the purpose and nature of the study & various physical examinations by the study to be carried out. I have been informed about the duration of the study and instructions to be followed during the study period.

I give full consent for being enrolled in the above study and I reserve my rights to withdraw from the study whenever I wish prejudice of my right to undergo further treatment at Santosh Hospital Ghaziabad U.P.

Signature of Attending Signature Of Patient

सं तोष मे डकिल कॉले ज और अस् पताल

सहमतपित् र

मै ं _____ पु त् र / पु त् री/धर् मपत् नी_____ नविासी_____ कोडॉक् टर Dr.Aarav Jawa द् वारा इस शोध कार् य के सम् बन् ध मे ं होने वाली हानि के बारे मे ं समझा दयिा है और मै ं इच् छा पू र् वक इस शोध कार् य जसिका शीर् षक "Clinical Study and management of secondary glaucoma" है और इस शोध कार् य के बारे मे ं समझा दयिा गया है l अतः मै ं उपरोक् त वषिय पर शोध कार् य मे ं हसि् सा ले ने के लएि सहमत हू ँ l मु झे समझा दयिा गया है की कसिी प् रतकि ू ल प् रतकि् रयिा के होने पर इलाज और मु आवजे की ज़मि् मे दारी जां च अधकिारी की है l मे री सभी जानकारी गु प् त रखी जाएगी और मु झे कसिी भी समय यह जां च छोड़ने का पू रा अधकिार है l मु झे समझादयिा गया है की इस जां च के दौरान मु झे कोई खतरा नहीं है l

हस् ताक् षर जां च अधकिारी के हस् ताक् षर

गवाह द् रभाष नं बर

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Introduction Glaucoma is a chronic, progressive optic neuropathy caused by a group of eye diseases that lead to damage to the optic nerve

head and a change in visual field. The most common risk factor generally acknowledged is a rise in intraocular pressure.1 Primary or secondary glaucoma are the two categories. Primary glaucoma is impacted by genetics. Secondary glaucoma occur when an underlying main ocular or systemic disease causes the IOP to increase.

Quite a few times secondary glaucoma goes undiagnosed or even if it is diagnosed it's treatment varies with each type of glaucoma. Consequently, early detection and treatment are crucial to preventing vision deterioration. The main cause of permanent blindness in the globe is glaucoma. According to WHO over 7.7 million persons with glaucomatous optic neuropathy are thought to exist in the globe.2 By 2040, it is estimated that there will be 111.8 million cases of glaucoma worldwide.3

In a recent study done in Aligarh of 680 total patients overall prevalence of glaucoma was found to be 5%, of which secondary glaucoma was 1% .4 Secondary glaucoma is divided into secondary open angle and secondary angle closure and these are further divided by basis of different mechanisms of causing secondary glaucoma

According to a study of 7079 patients conducted in Nepal on Glaucoma patients, 528 had secondary glaucoma i.e. 7% of the total cases. Out of these the most common was Lens-induced 33% then came Neo-vascular 20% followed by steroid-induced 16%, Trauma-induced 14%, Post-Vitrectomy Sx 3%, due to Uveitis 2%.5

Sometimes the treatment can also lead to further complications, as the side effect of corticosteroid usage as well as the disease itself, patients with uveitis are more likely to have a rise in intraocular pressure.6 Hence the treatment has to be carefully monitored.

Therapeutic modalities differ from one variety to another and what treatment is advised in one may not be the ideal treatment in the other type of sec.glaucoma.

In pigmentary dispersion syndrome which is a type of secondary open angle glaucoma, characterised by Trabecular Meshwork pigmentation, mid-peripheral radial iris transillumination abnormalities, and corneal endothelial pigment depositions on the posterior side of the cornea.7Lazer P.I. is considered a treatment modality and also has a protective role against rise of IOP over the coarse of 10yrs.8